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IMAGES IN CARDIOLOGY

Catheter Ablation of Incessant Ventricular Tachycardia in a Patient With Coronary Artery Disease

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A 67-year-old male with known coronary artery disease was referred to our hospital for catheter ablation of incessant ventricular tachycardia (VT). Transthoracic echocardiography revealed severe wall motion abnormalities of the left ventricle along with an apical aneurysm. Left ventricular voltage mapping showed a region with low voltage (<1.5 mV) at the left ventricular apex. Propagation mapping revealed a macro-reentry circuit around the apical aneurysm. Mid-diastolic potentials were recorded during the VT (**Fig**. 1, left panel, arrows), while entrainment mapping was excellent. The first radiofrequency energy application terminated the tachycardia. A circumferential lesion around the aneurysm was finally performed (**Fig**. 1, right panel, red dots). Ventricular tachycardia became non-inducible, and the patient is free from arrhythmic events during the last six months.

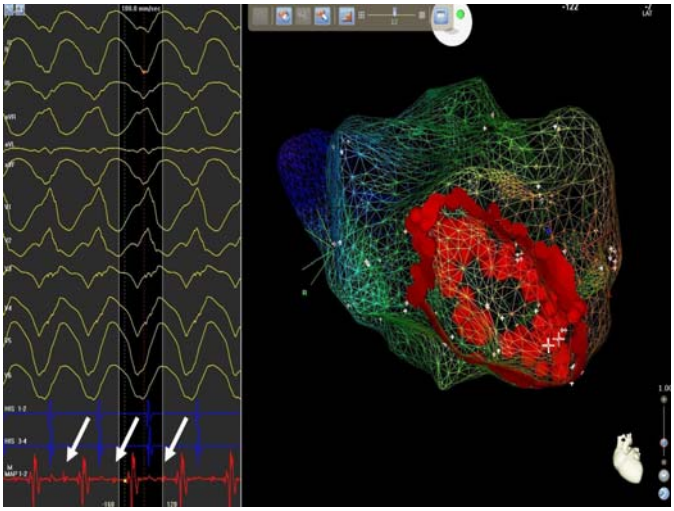

Figure 1. Surface ECG and intracardiac electrograms are depicted on the left panel and electroanatomical mapping on the right panel (see text for discussion)

Cardiology News /Recent Literature Review

Ector Anninos, MD, Spyridon Koulouris, MD, Antonis S. Manolis, MD

The **TCT** Annual Conference will be held in San Francisco 7-11/11/2011

The **AHA** Annual Scientific Sessions are scheduled for 12-16/11/2011 in Orlando

The next **ACC** Annual Meeting is slated for 24-27/3/2012 in Chicago

The **Athens Cardiology Update 2012** is slated for April 5-7, 2012

The **HRS** 33rd Annual Meeting will be held in Boston, 9-12/5/2012

The next **ESC** Annual Congress will be held in Munich, 25-29/8/2012

Incidence and Predictors of ICD Therapy in Patients With Arrhythmogenic Right Ventricular Dysplasia/ Cardiomyopathy (ARVC) Undergoing ICD Implantation for Primary Prevention

Eighty-four patients with definite or probable ARVC underwent ICD implantation for primary prevention. After a follow-up of 4.7 ± 3.4 years, appropriate ICD therapy was delivered in 40 patients (48%). Predictors of such an intervention were proband status ($p < 0.001$), inducibility at electrophysiologic study ($p = 0.005$), presence of nonsustained ventricular tachycardia ($p < 0.001$), and Holter premature ventricular complex count >1,000/24 h ($p = 0.024$). In multivariate analysis, inducibility at electrophysiologic study and nonsustained ventricular tachycardia remained significant predictors. The number of these risk factors correlates with the incidence of ICD activation with the 5-year survival free of appropriate ICD therapy for patients with 1, 2, 3, and 4 risk factors being 100%, 83%, 21%, and 15%, respectively (Bhonsale A et al, *J Am Coll Cardiol* 2011; 58:1485-1496).

Exclusion of the Left Atrial Appendage with a Novel Device: Early Results of a Multicenter Trial

Seventy patients with atrial fibrillation or a $CHADS_2$ score greater than 2 undergoing elective cardiac surgery were eligible for concomitant AtriClip device (35, 40, 45, and 50 mm) insertion. Safety was assessed at 30 days, and efficacy of left atrial appendage exclusion was assessed at operation (by transesophageal echocardiography) and at 3-month follow-up (by computed tomography angiography or transesophageal echocardiography). Intraprocedural success reached 95.7% (67 of 70 patients). Although significant adverse events occurred in 34 of 70 patients (48.6%), none was related to the device and there was no perioperative mortality. At 3-month follow-up, 98.4% of the patients had successful left atrial appendage exclusion by computed tomography angiography or transesophageal echocardiography imaging (Ailawadi G et al, *J Thorac Cardiovasc Surg* 2011; 142:1002-1009).

SCN5A Mutations Associate With Arrhythmic Dilated Cardiomyopathy and Commonly Localize to the Voltage-Sensing Mechanism

In this study the role of the cardiac sodium ion channel SCN5A in the etiology of dilated cardiomyopathy (DCM) was examined. To achieve this, DNA samples from 338 individuals among 289 DCM families were screened for SCN5A mutations by sequence analysis. The authors identified 5 missense SCN5A mutations representing the 1.7% of DCM families. Three of these mutations are novel (E446K, F1520L, and V1279I) and two are already known ones (D1275N and R222Q). In the majority of the cases the mutation was located to the highly conserved homologous S3 and S4 transmembrane segments, suggesting the disruption of the voltage-sensing mechanism of this sodium channel as the underlying mechanism leading to DCM. Most of the SCN5A mutation carriers manifested supraventricular arrhythmia (13/15), sick sinus syndrome (5/15) atrial fibrillation (9/15), ventricular tachycardia (5/15), and conduction disease (9/15) (McNair WP et al, *J Am Coll Cardiol* 2011;57: 2160-2168.)